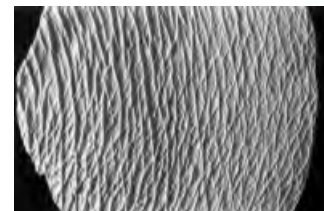


CLINICAL SNIPPETS

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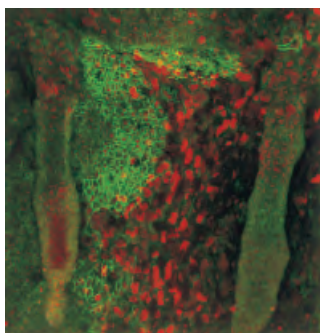
Predicting Skin Pattern

Shekar and coworkers, using 332 monozygotic and 488 dizygotic twin pairs at ages 12, 14 and 16, analyzed the cause of skin patterning, focusing particularly on genetics, sun exposure and skin color. Although sun exposure was a significant contributor to variation in skin pattern, it resulted in only 3.4% of variation in skin pattern at age 14. Genetic effects explained 86% of the variations at age 12, but this diminished to 62% in adulthood. Skin color explained 10.4% of variation in skin pattern at age 12; melanin content appears to provide a protective effect against skin pattern deterioration. *J Invest Dermatol* 125:1119–1129, 2005.



$\beta 1$ Integrin Deletion in Epidermis

López-Rovira and coworkers analyzed the consequences of deleting $\beta 1$ integrins in adult mouse epidermis. Dermo-epidermal splitting, inflammation, reduced proliferation, and hair follicle and sebaceous gland loss occurred in 30-day-old K5Cre $\beta 1$ -null mice, but these changes were not observed 30 days after $\beta 1$ integrin deletion in adult epidermis, and there were no changes in the hair follicle stem cell compartment. Their results highlight the context-dependent effects of $\beta 1$ integrin deletion and suggest that inflammation may be responsible for some of the K5Cre $\beta 1$ -null phenotype. *J Invest Dermatol* 125:1215–1227, 2005.



The Privileged Nail

The nail apparatus must combine effective anti-infection defenses and damage repair with safeguards against the loss of nail production as well as regenerating by autoaggressive/destructive immunity. Ito and colleagues, using cryospecimens of normal, uninflamed, uninfected human nail apparatus, determined the mechanism by which the nail immune system operates. The nail immune system differs strikingly from the skin immune system, but shows intriguing similarities to the hair follicle immune system, including the establishment of an area of relative immune privilege in the proximal nail matrix. Thus, there are morphological as well as immunological similarities between hair and nail. *J Invest Dermatol* 125:1139–1148, 2005.



Infrared Melts Raynaud's

One feature of scleroderma is Raynaud's phenomenon (RP), the vasospastic constriction of blood vessels in the fingers and toes. Foerster and coworkers examined the effect of IRA (infrared A) treatment on RP using as outcome variables the fingertip rewarming in response to cold challenge and a clinical activity score. IRA-mediated mild hyperthermia reduced the severity of scleroderma-associated RP and, if sustained treatment responses can be demonstrated in future research, may offer a valuable complementary treatment when drug side effects are limiting. *J Invest Dermatol* 125:1313–1315, 2005.

Itchy People

Yostipovitch and colleagues measured skin blood flow in 21 healthy volunteers at baseline and after exposure to thermal stimuli and scratching with a brush. Scratching reduced mean histamine-induced skin blood flow and itch intensity, and noxious heat (49°C) pain increased basal skin blood flow but reduced histamine-induced maximal skin blood flow and itch intensity. It appears that both histamine-induced skin blood flow and itch can be reduced when nerve fibers outside an itchy area are activated. Maybe new drugs to alter skin hyperemic responses will be developed as antipruritic agents, but these investigators say they've just scratched the surface of the causative mechanism! *J Invest Dermatol* 125:1268–1272, 2005.



Itchy Mice

Takakura and coworkers examined whether CpG oligodeoxynucleotide (ODN) could prevent atopic-dermatitis (AD)-like skin lesions in a mouse model. Sixteen of 26 NC/Nga mice did not exhibit dermatitis after CpG ODN was administered, but ten mice developed more severe lesions. Since CpG ODN also induced IFN- γ production, which inhibited the production of Th2 cytokines and ultimately led to a decrease in the serum IgE level, it may be that the suppression of Th2 cytokines may not completely prevent dermatitis and that IFN- γ may play a pathogenic role in some of these mice. *J Invest Dermatol* 125: 1156–1162, 2005.

